"EFFECT OF OCCUPATIONAL THERAPY INTERVENTIONS IN INSOMNIA AMONG PATIENTS WITH ANXIETY"

A PROJECT WORK SUBMITTED IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF OCCUPATIONAL THERAPY

(ADVANCED O.T. IN MENTAL HEALTH)

Submitted by

Reg. No. 411612052

JKK MUNIRAJAH MEDICAL RESEARCH FOUNDATION COLLEGE

OF OCCUPATIONAL THERAPY

KOMARAPALAYAM - 638183

Affiliated to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

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PRINCIPAL EXTERNAL EXAMINER

GUIDE INTERNAL EXAMINER

CERTIFICATE

This is to certify that the Project work entitled **"EFFECT OF OCCUPATIONAL THERAPY INTERVENTIONS IN INSOMNIA AMONG PATIENTS WITH ANXIETY"** is a bonafied compiled work carried out by **Reg. No. 411612052,** Final Year student, College of Occupational Therapy under JKK Munirajah Medical Research Foundation, Komarapalayam – 638183, in partial fulfillment for the award of Degree of **"Master of Occupational Therapy"** (Advanced O.T. in Mental Health) of The Tamil Nadu Dr. M.G.R. Medical University,Chennai-32. This work was guided and supervised by **Mr. T. JEGADEESAN. M.O.T., M.Sc.,(Psy)** at the **Department of Occupational Therapy**, JKKMMRF, Komarapalayam.

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ABSTRACT

OBJECTIVE

The purpose of the study was to evaluate the effect of occupational therapy intervention in insomnia among people with anxiety.

METHODS

Totally 30 subjects, 15 in experimental group and 15 in control group with age of 20 to 45 years participated in current study.

RESULTS

The mean of post-test was found to be 96 (control) and 62.93 (experimental) respectively. The calculated‘t’ value was obtained to be 3.785 with level of significance 0.05.Statistical significance is present in experimental group than control group with regard to effect of occupational therapy intervention with conventional therapy.

CONCLUSION

Occupational therapy intervention has statistically significant effect on the reduction of Insomnia.

KEY WORDS

Insomnia, Anxiety, Occupational therapy intervention.

INTRODUCTION

Insomnia is a serious health concern which result in poor day time functioning and work performance, people with insomnia have more medical problems than people without insomnia[29].The causes of sleep loss are multifactorial and it fall under two major, somewhat overlapping categories: lifestyle or occupational such as shift work, prolonged working hours, irregular sleep schedules, and sleep disorders such as Breathing-Related Sleep Disorders, narcolepsy, circadian rhythm disorders, Restless Leg Syndrome. Unfortunately, available epidemiological data are not sufficient to determine the extent to which sleep loss is caused by pathology versus behavioural components.

In the past years research has overturned the dogma that sleep loss has no health effects, apart from daytime sleepiness. Studies suggest that sleep loss may have wide-range of effects on the cardiovascular, endocrine, immune, and nervous systems, including Obesity in adults and children, Diabetes and impaired glucose tolerance, Anxiety symptoms, depressed mood andalcohol use, Even insomnia is associated with suicidal ideation among patients with depression, alcohol misuse or dependence, and posttraumatic stress disorder. The link between insomnia and suicide is evident across the life span, including adolescents and older adulthood.

Estimates of insomnia prevalence have varied widely, from 10% to 40%. According to National Institutes of Health 2005, insomnia has a prevalence of 10% if the definition necessitates daytime distress or impairment, given all the information available, the prevalence of insomnia symptoms may be estimated at 30% and specific insomnia disorders at 5% to 10%.

Sleep is an essential aspect of daily life for which occupational therapy services may be warranted, and there is a great need to expand the profession's understanding of sleep-related interventions[13].As an Occupational therapy practitioner it is important to analyse the day time consequence of Insomnia that include, Daytime sleepiness, fatigue, depressed mood, lack of energy, impaired cognition, memory problems, irritability, psychomotor dysfunction, and decreased alertness and concentration. These consequences affect overall Activities of daily living (ADL) and productivity of an individual. ADL and productivity are the important areas of occupational therapy practice.

Cognitive behavioural therapy for insomnia (CBT-I) is effective at reducing insomnia symptom severity,including in populations with comorbid conditions. The underpinnings of CBT-I flow from the application of both operant and classical conditioning paradigms in the form of stimulus control instruction, the focus on sleep-interfering behaviours in the form of sleep hygiene, the recognition of and focus on reducing the hyperarousal features of insomnia.

Even though there is a large population is suffering from the consequences of insomnia which directly affects the daily life and work performance of an individual, very less occupational therapy practitioner address these problems. As an occupational therapy practitioner it is important to have a deep understanding of sleep and sleep related problems hence this study has been initiated.

OPERATIONAL DEFINITION

INSOMNIA:

"An insomnia disorder is defined as a subjective report of difficulty with sleep initiation, duration, consolidation, or quality that occurs despite adequate opportunity for sleep, and that result in some form of daytime impairment".

ANXIETY:

Anxiety is an emotion characterized by feelings of tension, worried thoughts, distributed sleep and physical changes like increased blood pressure. People with anxiety disorders usually have intrusive thoughts and concerns. They avoid certain situations out of worry.

NEED OF THE STUDY

The consequences of poor sleep are multifaceted and can result in decreased academic and work performance. Even though sleep is an essential aspect of daily living there are less research happened in occupational therapy servicerelated to sleep. So there is a need to explore the significance of occupational therapy intervention in sleep.

AIM

The aim of the study is to find out the effect of occupational therapy interventions in insomnia among person with anxiety.

OBJECTIVES

- To assess the level of insomnia among person with anxiety.
- To evaluate the effect of occupational therapy intervention in Insomnia.
- To compare the effect of conventional therapy and occupational therapy techniques.

HYPOTHESIS

ALTERNATE HYPOTHESIS

- Occupational therapy technique will have a significant effect on insomnia.

NULL HYPOTHESIS

- Occupational therapy technique will have no significant effect on insomnia.

PHYSIOLOGY OF SLEEP/WAKE TRANSITION:

By definition, sleep is the alternate condition of wakefulness[47] the state of wakefulness is primarily driven by the activity of specific cell groups of the ascending reticular arousal system (ARAS) located in the brainstem. The Ascending Reticular Activating System is composed by two branches (Figure 1). The first originates from the acetylcholine nuclei (i.e. the pedunculo-pontine and laterodorsal tegmental nuclei) and innervates the thalamocortical neurons, whereas the second originates from monoaminergic and glutamatergic cell groups in the brainstem, bypasses the thalamus, and activates neurons in the lateral hypothalamic area and basal forebrain[59].

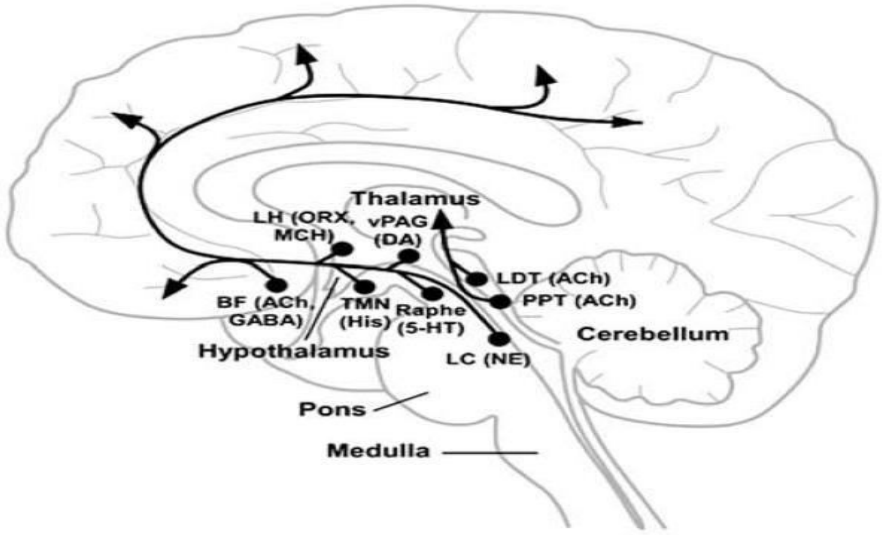

Figure 1: Cholinergic neurons innervate the thalamus, whereas monoaminergic and glutamatergic neurons extended to the hypothalamus, basal forebrain, and cerebral cortex. The orexin neurons in the lateral hypothalamus indirectly reinforce the brainstem arousal pathways and directly excite the cerebral cortex and basal forebrain. Basal forebrain (BF) : Gamma-Amino Butyric Acid(GABA), Ach: acetylcholine); raphe:, dorsal raphe nucleus (5-HT: serotonin); LC: locus coeruleus (NA: norepinephrine); LDT: laterodorsal tegmental nucleus (Ach); LH: lateral hypothalamus (ORX: Orexin; MCH: melanin-concentrating hormone), PPT: pedunculopontine tegmental nucleus (Ach); TMN: tuberomammillary nucleus (His: histamine); vPAG: ventral periaqueductal gray (DA: dopamine). (adapted from Saper, et al.[59]).

Wakefulness is further promoted by the orexin/hypocretin neurons in the lateral hypothalamus and the acetylcholine neurons of the basal nuclei.

Sleep, however, as correctly identified 80 years ago by Von Economo[65], is promoted by neurons in the lateral hypothalamus. More specifically, neurons in the ventral lateral preoptic area (VLPO) inhibit the monoaminergic cell groups in the hypothalamus and brainstem using galanin and Gamma-Amino Butyric Acid neurotransmitters. However, during the wake state, theVentral Lateral Preoptic Area neurons are themselves inhibited by the Ascending Reticular Arousal System (ARAS). This reciprocal inhibitory state has been called the "flip-flop" switch[59], which allows the rapid transition from a wake condition to a sleep state and vice versa. Thus, the sleep/wake transition is regulated by two mutual inhibition systems that cyclically suppress each other.

These systems seem to be hierarchically influenced by both the homeostatic and the circadian regulation of sleep[59], which constitute the basis of the two-process model of sleep regulation[18].According to this model, the sleep-wake alternation depends on a sleep-dependent homeostatic process and a sleep-independent circadian process. The homeostatic drive depends on the previous waking time: the propensity to sleep increases during wake and dissipates during sleep. The circadian process is independent of the sleep pressure and modulates sleep pressure as a function of exogenous (e.g. light) and endogenous rhythms (e.g. biological clock). Sleep is then the result of the interaction between these two processes.

This model has provided accurate predictions regarding wake and sleep behaviour in different experimental setting and in free-living environments[14]. Recently adenosine, a by-product of brain energy metabolism (i.e. the final results of

a double ATP breakdown) has been proposed as the physiological accumulator of the need for sleep[54]. During wakefulness, adenosine accumulates in the basal forebrain as a consequence of ATP consumption, whereas during sleep its levels decrease in the cortex, basal forebrain, hypothalamus, and brainstem[17]. It has been hypothesized that slow wave activity is the physiological marker of this adenosine down-regulation[43]. Adenosine seems to disinhibit the ventral lateral preoptic area neurons, and inhibits hypocretin or orexin and the basal forebrain neurons.[17] Interestingly, caffeine is as an adenosine receptor antagonist, caffeine prevents sleep by binding to adenosine receptors.

SLEEP ARCHITECTURE:

Sleep is not a unique phenomenon, but a structured sequence of events that follows a regular, cyclic program. It is organized in different phases (or stages), each one characterized by a specific pattern of tonic and phasic physiological activity. Sleep is composed of two principal states, namely Non-rapid eye movement (NREM) or Synchronized sleep and Rapid eye movement (REM), also called desynchronized sleep or paradoxical sleep. These two sleep states cyclically alternate during sleep depending on several factors such as, time of day, temperature, sleep pressure, and environmental conditions. Non-rapid eye movement sleep is further divided in three stages (N1, N2, N3 or slow wave sleep, SWS) according to the amplitude and frequency of the electroencephalographic (EEG) activity.

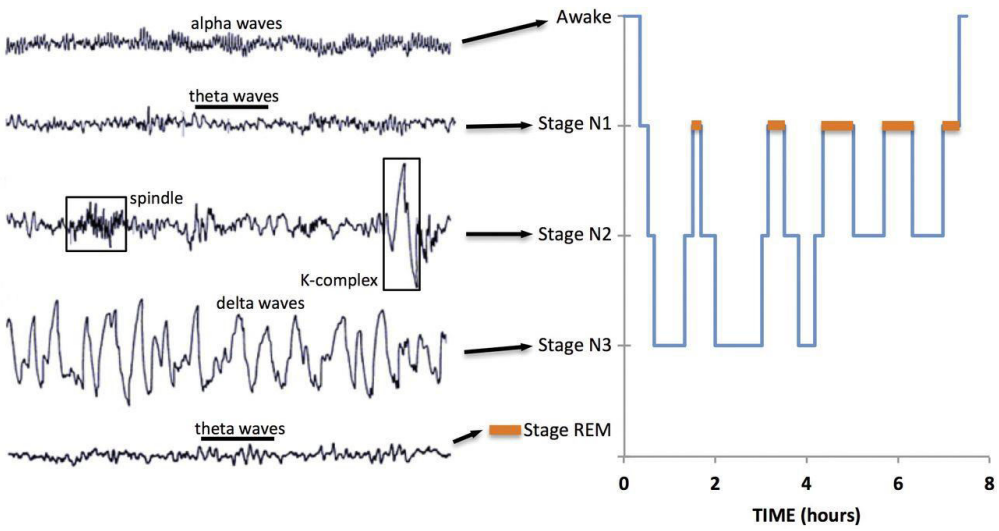

Figure 2

During nocturnal sleep there are 4-6 cyclical alternations of non-rapid eye movement and rapid eye movement phases. In the first cycle, sleep onset usually occurs in N1 sleep which is followed by N2 and then SWS. Afterwards, sleep typically becomes lighter, with a brief period of N2 or N1 preceding the onset of rapid eye movement. The end of rapid eye movement is indicated by a short awakening from sleep and/or a body movement or a transition back to N2 sleep. Each sleep cycle lasts 90-110 minutes and differs from the others. N1 sleep usually occurs at sleep onset and as a transitional state across the night, representing about 2 to 5 % of nocturnal sleep. N2 sleep is the more stable stage, occurring constantly through the night and constituting about 45 to 55 % of sleep. SWS, which represent about 20 to 25 % of total nocturnal sleep, is predominant in the first half of the night, often occurring only in the first two cycles. On the other hand, the first rapid eye movement episode is usually short (1-5 min) but then becomes longer across cycles, becoming predominant in the second half of the night, amounting to 20-25% of the sleep.[22]

INSOMNIA:

Insomnia is the most common sleep disorder which affects about 10–15% of the populationand is defined as difficulty in falling asleep, maintaining sleep or non-restorative sleep (American Academy of Sleep Medicine, 2005; American Psychiatric Association, 2000). In addition to nocturnal symptoms, daytime consequences are frequently reported by insomniacs, in particular, increased daytime sleepiness, fatigue, mood disturbance, exhaustion, dysphoria which generates significant distress (American Academy of Sleep Medicine, 2005), functional and cognitive impairment and reduced quality of life.

The core symptoms of insomnia, specified in major disease and sleep disorder classification manuals (ICSD-3, 2014; DSM-V, 2013; ICD-10, 1992), correspond to difficulties with initiating and/or maintaining sleep, or non-restorative sleep (i.e. poor quality sleep). To achieve disorder 'status', sleep disturbance must not be simply a function of restricted sleep opportunity (i.e. curtailment), or environmental perturbation (such as noise, bed partner snoring etc.). Importantly, the diagnosis of insomnia disorder is made only when impairment in daytime functioning is present, which is linked (attributionally) to night-time sleep difficulties. Daytime impairments may be measured with reference to isolated symptoms, such as fatigue and but also to more global dysfunction; for example, in areas of social and occupational functioning (DSM-V, 2013). One additional marker of insomnia severity refers to the frequency and length (persistence) of insomnia symptoms, which, for an insomnia diagnosis, is usually set at greater than or equal to three nights per week, being present for at least a one month period.

It has been shown that insomnia is linked with absenteeism (at least twice in workers with insomnia than workers without insomnia,[37] accidents,decreased productivity and efficiency at work and decreased job satisfaction. [41]

Epidemiological data suggest that about one-third of the general population suffer from symptoms of insomnia,[52] with literature showing a prevalence ranging from 5% to 40% depending on different definitions. When consensus criteria are applied, the reported prevalence is between 20% and 25% of adults.[52] Primary insomnia, which is an insomnia not due to other medical conditions or sleep disorders, seems to account for 3% of those diagnosed with insomnia. Insomnia is more prevalent in females than males and symptoms of insomnia increase with aging.[62] Insomnia is a very common sleep disorder already in early adulthood, with incidence remaining stable across adulthood.

Furthermore, insomnia is associated with depression and anxiety[48]and seems to be associated with an increased risk for cardiovascular diseases. In addition, it has been observed an association between insomnia and mortality. Other evidence reported elevated heart rate and altered heart rate variability (HRV) in insomnia patients that are known to be risk factors for cardiovascular disease and mortality.[35]

Although the pathogenesis of primary insomnia is still unknown, nocturnal symptoms as well as diurnal complaints in insomniacs may be attributable to a chronic state of hyperarousal, i.e. a condition of elevated physiological activation that affects somatic, cortical, and cognitive functioning throughout the day as well as at night, leading to nocturnal and diurnal symptoms.

Indeed, night time and daytime studies of insomniacs show decreased nocturnal production of melatoni, high levels of cortisol and ACTH, increased body

metabolic rate, basal temperature, heart, blood pressure, muscular tone and electrodermal activity; decreased pre-ejection period, elevated high frequency EEG activity in both rapid eye movement and non-rapid eye movement sleep, abnormal intracortical excitability, and increased subjective perception of hyperarousal as assessed by questionnaires.[55]

Pharmacotherapy

A number of pharmacological agents exists for the treatment of insomnia symptoms, most of which primarily work via their agonistic effects on gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the central nervous system (CNS). Specifically, the most common class of hypnotics, benzodiazepines (e.g. temazepam), and the newer non-benzodiazepine receptor agonists (zolpidem, eszopiclone; BZRAs or 'z drugs') modulate GABAA receptors, facilitating the inhibitory effects of gamma-aminobutyric acid on overall CNS arousal, while similarly inhibiting norepinephrine activity. Benzodiazepine receptor agonists are considered more effective due to their greater selectivity for the alpha 1 sub-unit, thus enhancing activity in terms of sedation and limiting more generic effects involved in the interaction with other subunits (Nutt & Stahl, in press). These compounds also tend to have shorter half-lives than the original benzodiazepines, reducing the likelihood of carry-over effects the next day; though available data on side-effects and comparable efficacy do not allow for clear conclusions to be made.[31]

Although demonstrating effectiveness (moderate to large effects) in terms of improving major indicators of sleep continuity and quality in those with insomnia, positive effects of Benzodiazepine receptor agonists have yet to be reliably demonstrated beyond active administration. Indeed, as it stands, there is not enough

evidence to suggest or recommend that Benzodiazepine receptor agonists are useful for the long-term management of chronic insomnia beyond four weeks of treatment (NIH state-of-the-science conference statement, 2005). Some recent studies report improvements during extended/intermittent use of hypnotics over 6 and 12 month periods but again no adequate data exist indicating maintained benefits long after treatment cessation. Concerns about long-term hazardous side-effects and tolerance issues strongly argue against long-term prescription: safety and efficacy must be documented.

Cognitive Behavioural Therapy for Insomnia (CBT-I)

Given that maladaptive behaviours and cognitive processes are thought to underlie the maintenance of insomnia, it is intuitive that therapy targets these factors directly.[58] Cognitive behavioural therapy for insomnia (CBT-I) is an evidence-based treatment modality, containing a number of supported techniques to improve sleep. Practice parameters set out by the American Academy of Sleep Medicine (AASM) recommend and endorse the following single components: paradoxical intention therapy, stimulus control therapy, sleep restriction therapy, progressive muscular relaxation, and biofeedback.[25] In addition, two multicomponent CBT approaches are also supported. Indeed, most outcome research has focused on multi-component, multisession CBT interventions

Combination therapy:

Combination therapy involves prescribing both cognitive behavioural therapy for insomnia (CBT-I) and a medication, usually for six to eight weeks.The medication is then tapered off or to an as-needed schedule, while continuing the CBT-I.

Taken together, the evidence indicates that CBT-I alone, drug therapy alone, and combination therapy all improve measures of insomnia (eg, wake time after sleep onset) within weeks of initiating the therapy. Continuing CBT-I alone after the completion of initial therapy appears to be the best option for maintaining improvement long-term. CBT-I also increases the likelihood that the medication can eventually be tapered. If sleep restriction therapy is combined with hypnotic medication, clinicians should be aware that the combination of chronic partial sleep deprivation and medication hangover could significantly increase daytime sleepiness and behavioural risk. The evidence is insufficient to justify combination therapy as routine initial management for insomnia patients. Many patients will improve with CBT-I alone, without pharmacologic therapy.

Follow-up

If the treatment is successful, patients will report both improved sleep at night and improvement of daytime deficits. Discontinuation of the medication should be considered in any patient who is receiving pharmacologic therapy alone or combination therapy.

Patients who have little improvement during the initial trial of cognitive behavioural therapy, pharmacologic therapy, or combination therapy may have other causes of poor sleep. Adherence with the prescribed therapy should be confirmed and then additional diagnostic evaluation performed.

GENERALIZED ANXIETY DISORDER:

GAD often presents through hyper-vigilance, excessive worrying, muscle tension, irritability, fatigue, and restlessness. These key features are difficult for the

patients to manage and control, keeping them from completing daily tasks (American Psychiatric Association, 2013). To be diagnosed with generalized anxiety disorder through the DSM-V, a person must exhibit three or more of the six symptoms listed in the DSM those are

A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

B. The individual finds it difficult to control the worry.

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months); Note: Only one item is required in children.

    1. Restlessness or feeling keyed up or on edge.
    2. Being easily fatigued.
    3. Difficulty concentrating or mind going blank.
    4. Irritability.
    5. Muscle tension.
    6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep).

D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).

F. The disturbance is not better explained by another mental disorder (e.g., anxiety or worry about having panic attacks in panic disorder, negative evaluation in social anxiety disorder [social phobia], contamination or other obsessions in obsessive-compulsive disorder, separation from attachment figures in separation anxiety disorder, reminders of traumatic events in posttraumatic stress disorder, gaining weight in anorexia nervosa, physical complaints in somatic symptom disorder, perceived appearance flaws in body dysmorphic disorder, having a serious illness in illness anxiety disorder, or the content of delusional beliefs in schizophrenia or delusional disorder).

Causes of Generalized Anxiety Disorder (GAD)

It is thought differences in the following neurotransmitters play a role in causing GAD:

- Serotonin
- Dopamine
- Norepinephrine
- Gamma-aminobutyric acid (GABA)

It is these chemicals that are altered by antidepressants, some of which are effective treatments for generalized anxiety disorder. Abnormal levels of other chemicals, like peptides and hormones, may also partially cause generalized anxiety

disorder. MRI scans have revealed that some structures of the brain are changed in some anxiety disorders.Impaired cognitive functioning also appears to be tied to generalize anxiety disorder in both children and adults. It may also as a result of the following.

- Early traumatic experiences such as a parent's death
- Chronic experiences of fear
- Chronic feelings of helplessness
- Abnormal hormones, possibly due to stress, prenatally

Pharmacotherapy:

Antidepressants, particularly the selective serotonin reuptake inhibitors (SSRIs), are widely used to treat and prevent a variety of anxiety disorders. Examples of SSRIs that are commonly used to treat chronic anxiety include citalopram (Celexa), escitalopram (Lexapro), fluoxetine (Prozac), paroxetine (Paxil), and sertraline (Zoloft). The antidepressants duloxetine (Cymbalta) and venlafaxine (Effexor), SNRIs (serotonin and norepinephrine reuptake inhibitors) which act on the brain chemicals serotonin and norephinephrine, and some of the tricyclic antidepressants like imipramine (Tofranil), may also help. Antihistamines (such as hydroxyzine) and beta-blockers (such as propranolol) can help mild cases of anxiety as well as performance anxiety, a type of social anxiety disorder. Antidepressants such as SSRIs or SNRIs or tricyclics need to be taken daily whether or not anxiety has on that particular day. Antihistamines or beta-blockers are usually taken only when needed for anxiety, or immediately before an anxiety-provoking event (for example, taking propranolol shortly before giving a speech). Finally, certain anticonvulsant medicines, such as

gabapentin (Neurontin) and pregabalin (Lyrica), are also beginning to show value in treating some forms of anxiety in initial research studies acute anxiety. The most prominent of anti-anxiety drugs for the purpose of immediate relief are those known as benzodiazepines; among them are alprazolam (Xanax), clonazepam (Klonopin), chlordiazepoxide (Librium), diazepam (Valium), and lorazepam (Ativan). They have drawbacks: Benzodiazepines sometimes cause drowsiness, irritability, dizziness, memory and attention problems, and physical dependence. Nonetheless, in recent decades they have largely replaced barbiturates because they tend to be safer if taken in large doses.

Another anti-anxiety drug is busprirone (Buspar). It has fewer side effects than the benzodiazepines and is not associated with dependence. Buspar, however, can have its own side effects and may not always be as effective when a person has taken benzodiazepines in the past.

Cognitive behavioural therapy:

Cognitive behavioural therapy (CBT) is based on evidence that shows that persons with generalized anxiety disorder (GAD) engage in overestimations and catastrophizing of negative events; show limited confidence in problem solving; require additional evidence before making decisions; have a low tolerance of uncertainty, an iterative problem-solving style, worry about worry, and numerous behavioral and cognitive strategies that may actually be counterproductive and help maintain the self-perpetuating cycle of worry.

Specific techniques: CBT is a multimodal intervention for GAD, including patient education, self-monitoring, relaxation training, cognitive restructuring,

imagery exposure, exposure to anxiety provoking situations, and relapse prevention. These techniques are described below.

Education — Treatment begins with education on:

- Informing and correcting misconceptions regarding anxiety, worry, and associated symptoms
- Causative factors of pathological worry and anxiety
- A model of factors that perpetuate GAD
- The treatment plan and rationale (ie, symptoms of GAD will subside by using evidence-based and coping oriented thinking, by dealing directly with anxiety provoking images and situations, and by learning to relax)

Much of this information is integrated in presenting how a pathological cycle of worry and anxiety develops and is maintained in patients' lives.

Self-monitoring: Self-monitoring is introduced in the first treatment session and continues throughout the entire treatment. Learning to observe their reactions from an objective standpoint encourages the patient's development as a personal scientist and increases his or her accuracy in self-observation. Self-monitoring allows patients to chart their progress in therapy.

Relaxation training: Relaxation training can be particularly meaningful for GAD patients as they often experience elevated muscle tension and reduced flexibility of autonomic functioning. Relaxation training consists of progressive muscle relaxation (after brief deliberate tension) of all muscle groups of the body in a systematic manner, beginning with 16 muscle groups, and then condensing to 8

muscle groups, and 4 muscle groups. Relaxation training ends with cue-control relaxation, where patients cue themselves to relax by simply repeating a word (such as "relax") that has been repeatedly paired with relaxation phases during the preceding weeks of progressive muscle relaxation training. Cue-control relaxation is then used as a coping skill for practicing exposure to anxiety-producing images or situations (also referred to as "applied relaxation"). Breathing exercises, such as slow, diaphragmatic breathing, may be incorporated into relaxation training.

Cognitive restructuring: Cognitive restructuring is a set of skills for identifying and modifying misappraisals that contribute to anxiety, including:

- Patients are shown how anxiety and maladaptive behaviors are generated by overly-negative interpretations of events.

- Patients are helped to identify errors in thinking (eg, overestimating the probability or valence of negative events) and rigid rules or beliefs that underlie dysfunctional thought patterns.

- Patients are encouraged to use an empirical approach to examine the validity of thoughts by considering all of the available evidence.

Therapists use Socratic questioning to help patients make guided discoveries and question their anxious thinking. Patients then generate alternative interpretations or "hypotheses" to situations with the help of additional evidence gathered in behavioral practices in anxiety-provoking situations. As an example, a person who typically avoided taking on new responsibilities due to worries about making mistakes was encouraged to take on new responsibilities, to gather evidence on what happens subsequently. He or she learned that mistakes were less frequent than anticipated and

did not have negative consequences. Underlying beliefs (eg, “I am incompetent”) are postulated to change with the patient’s accrual of evidence that challenges his or her negative thoughts.

Imagery exposure: Imagery exposure is designed to help patients tolerate negative affect and autonomic arousal associated with fearful images that they often attempt to avoid through worry. Patients generate hierarchies of fear images related to two or three main areas of worry and are led through systematic exposure to these images. When anxiety elicited by an image is reduced to a mild level, then patients progress to the next image on the hierarchy. Two main versions of imagery exposure have been developed

- In one version, patients imagine a worst case scenario for 25 to 30 minutes, and then generate alternative outcomes to the scenario. This approach has been shown to be effective for GAD as a standalone treatment in a small randomized trial.

- The second version, self-controlled desensitization, involves utilization of cognitive restructuring and relaxation skills during imagery exposure to anxiety-provoking situations. It has been incorporated into CBT in a number of studies.

Exposure to anxiety-provoking situations: This technique involves repeated exposure to situations that are avoided or engaged in with excessive preparation or checking. Patients generate a hierarchy of situations or activities. Examples include allowing children to have sleep overs, family vacations, arriving on time (instead of excessively early) at scheduled appointments, taking on responsibilities, or saying ‘no’ to requests. Patients rehearse cognitive restructuring and relaxation coping skills

in session. Subsequently, they practice using these techniques to manage anxiety in situations that occur between sessions.

Relapse prevention : A final step in CBT is relapse prevention, in which patients are informed that recurrences of worry, anxiety or avoidance behavior are likely to occur in the future. They are encouraged to view such recurrences as lapses rather than failure, and to reapply their coping skills and reinstitute their practice of exposure to images of negative outcomes and anxiety-provoking situations.

Psychodynamic therapy:

In psychodynamic approaches to GAD, treatment typically focuses upon core conflictual relationship themes. Emphasis is placed upon a positive therapeutic alliance to provide a corrective emotional experience to offset insecure attachment.

Emotional regulation therapy: Emotional regulation therapy incorporates components of CBT such as psychoeducation and self-monitoring, as well as interventions that address emotion regulation (deficits prominent in GAD), emotional avoidance, and interpersonal difficulties.

Mindfulness: Mindfulness involves the non-judgmental observation of moment to moment experiences. Acceptance and Commitment Therapy combines mindfulness with acceptance of internal states and orientation of actions towards valued goals. Although there are some similarities between an ACT approach and a CBT approach, ACT does not involve any form of cognitive restructuring (ie, identifying, challenging, and replacing negative thinking with more realistic thinking) or any attempt to change or correct somatic dysregulation (eg, relaxation training). A randomized clinical trial compared ACT to applied relaxation in patients with GAD.

Aaron M Eakman et al (2017)[13]

They conducted a pilot study on restoring effective sleep tranquillity (REST). The participants were United States post-9/11 veterans with service-connected injuries, university students, and had self-reported sleep disturbances. REST was a multi-component cognitive behavioural therapy for insomnia intervention consisting of seven sessions of group therapy and eight 1:1 sessions delivered by occupational therapists. Indicators were supportive of feasibility, including reduced sleep difficulties, reduced nightmares, fewer dysfunctional sleep beliefs and greater ability to participate in social roles, along with trends towards improved satisfaction with participation and reduced pain interference.

ZubiaVeqar et,. Al (2014)[70]

The purpose of this study was to establish test- retest reliability, validity and internal consistency of Pittsburgh insomnia rating scale among Indian population. Twenty five subjects were randomly chosen from the screened population of poor sleepers. The study findings suggest that Pittsburgh insomnia rating scale has excellent internal consistency, test-retest reliability and good validity for university population of poor sleepers in India. It is an important first line of assessment scale for screening of sleep problems.

Christi S. Ulmer et,.al (2011)[26]

They conducted a pilot study on Multi-Component Cognitive-Behavioral Intervention for Sleep Disturbance in Veterans with PTSD. They included twenty-two veterans for the study, The findings demonstrate that an intervention targeting trauma-

specific sleep disturbance produces large short-term effects, including substantial reductions in PTSD symptoms and insomnia severity.

Yong Woo Kim et,. Al (2009)[68]

The objective of his study was to examine the effectiveness of newly developed Mindfulness-based cognitive therapy program as an adjuvant to pharmacotherapy in the treatment of patients with panic disorder or generalized anxiety disorder. Patients with panic disorder or generalized anxiety disorder were assigned to either MBCT or an anxiety disorder education (ADE) program for a period of 8 weeks. As a result the obsessive-compulsive and phobic subscales showed significantly more improvement in the Mindfulness-based cognitive therapy group. However, no significant improvement was observed in the Mindfulness-based cognitive therapy group versus the anxiety disorder education group in terms of the somatization, interpersonal sensitivity, paranoid ideation, or psychoticism subscale scores.

Jason C. Ong (2008)[40]

This study was designed to evaluate the efficacy of mindfulness meditation for the treatment of chronic insomnia. It was a randomized controlled trial with fifty-four adults with chronic insomnia. The result of the study suggests that mindfulness meditation appears to be a viable treatment option for adults with chronic insomnia and could provide an alternative to traditional treatments for insomnia.

Dag Neckelmannet.al.,(2007)[28]

This study was aimed to find out the relationship of insomnia to the development of anxiety disorders and depression in a population-based sample. They

have done a Cohort study based on data from two general health surveys of the adult population. After analysing the data it was prominent that insomnia may be a trait marker for individuals at risk for developing anxiety disorders.

Daniel J. Taylor, et al (2007)[29]

The study was aimed to determine the comorbidity of insomnia with medical problems. A Cross-sectional and retrospective study was conducted with Community-based population of 772 men and women, aged 20 to 98 years old. This study demonstrates significant overlap between insomnia and multiple medical problems, the study also suggest that people with insomnia had greater depression and anxiety levels than people not having insomnia and were 9.82 and 17.35 times as likely to have clinically significant depression and anxiety, respectively.

BørgeSivertsen, et al (2006)[19]

The study was aimed to examine short- and long-term clinical efficacy of cognitive behavioural therapy (CBT) and pharmacological treatment in older adults experiencing chronic primary insomnia. The study was a randomized, double-blinded, placebo controlled trial of 46 adults, outcome of this study suggest that interventions based on CBT are superior to zopiclone treatment both in short- and long-term management of insomnia in older adults.

Andrew Green et al (2005)[15]

The study was A Cognitive-Behavioural Group Intervention for People with Chronic Insomnia, the nonpharmacological management of chronic insomnia led to the establishment of a treatment group in which occupational therapists have been involved. The outcome data from seven groups are presented. Sleep diaries indicate a

modest improvement in total sleep time, with further improvement at 3-month follow-up. Participant feedback shows that people with insomnia value expert advice and support as well as meeting others with similar problems.

Catherine D. Jefferson (2005)[23]

This study was designed to assess selected aspects of sleep hygiene from a population-based sample of individuals with insomnia compared to age- and sex-matched controls. They included a population of 258 individuals with age group of18 to 65 years. The study suggest that insomniacs do engage in specific poor sleep hygiene practices, such as smoking and drinking alcohol just before bedtime. These particular aspects of sleep hygiene may be important components that exacerbate or perpetuate insomnia.

Ce´lyne H. Bastienet al (2004)[24]

This study was designed to compare the effect CBT delivered through Individual Therapy, Group Therapy, and Telephone Consultations. They give CBT for Forty-five adults with primary insomnia and found out that CBT was effective in improving sleep parameters with all 3 methods of treatment implementation, and there was no significant difference across methods of implementation.

Maurice M etal (2001)[42]

The study was designed to determine the role of activity status and social life satisfaction on the report of insomnia symptoms and sleeping habits. The study used a large population of 13,057 subjects age 15 and older. In this study they found out that Insomnia symptoms were reported by more than one-third of the population age 65 and older. Multivariate models showed that age was not a predictive factor of

insomnia symptoms when controlling for activity status and social life satisfaction. The level of activity and social interactions had no influence on napping, but age was found to have a significant positive effect on napping.

Colin A. Espie et al (2001)[27]

This study specifically investigated the clinical effectiveness of CBT delivered by Health Visitors (primary care nurses) trained as therapists. One hundred and thirty-nine insomniacs (mean age 51 yr) were randomised to CBT or Self-Monitoring Control (SMC) in a controlled trial. Superiority of CBT over SMC in substantially reducing sleep latency and wakefulness during the night. CBT-DEF replicated similar effects and maintained improvement was observed in both groups one year later. Furthermore, total sleep increased significantly during follow-up and 84% of patients initially using hypnotics remained drug-free. Results suggest that CBT administered by Health Visitors offers a clinically effective treatment for insomnia.

Spielmanetal (1987)[63]

In this study they assigned thirty-five patients, with a mean age of 46 years and a mean history of insomnia of 15.4 years. And were treated initially by marked restriction of time available for sleep, followed by an extension of time in bed contingent upon improved sleep efficiency, At the end of the 8-week treatment program, patients reported an increase in total sleep time as well as improvement in sleep latency, total wake time, sleep efficiency, and subjective assessment of their insomnia. Improvement remained significant for all sleep parameters at a mean of 36 weeks after treatment in 23 subjects participating in a follow-up assessment.

Robin puderetal (1983)[56]

This study was designed to find out Short-Term Stimulus Control Treatment of Insomnia in Older Adults. They did a 4-week stimulus control treatment to 16 ambulatory, noninstitutionalized older adults with sleep onset insomnia of which nine subjects received immediate treatment, and 7 received delayed treatment. This study confirms that short-term stimulus control therapy is effective in decreasing sleep-onset among insomnia in older adults.

STUDY DESIGN:

Quasi Experimental Design.

Experimental group

Pre-test → Occupational therapy intervention + conventional therapy → Post test

Control group :

Pre-test → Conventional Therapy only → post test

SAMPLE SIZE

Total consecutive samples of 30 subjects were taken for this study. The subjects were divided into two groups as experimental and control group. The control group consist of 15 subjects, and experimental group consist of 15 subjects.

Table 1

Group	AGE	SEX	
		Male	Female
Control Group	Mean: 32.2	9	6
Experimental Group	Mean: 32	10	5

SAMPLING TECHNIQUE

Convenient sampling technique was adopted.

STUDY PLACE

Vivekananda Health Centre, Erode.

DURATION OF THE STUDY

Duration of the study is one year

SELECTION CRITERIA

Inclusion criteria

- Participants with Insomnia between ages 20 to 45 years.
- Participants with onset of illness from 3months to 1 year.
- Both male and females
- Participants should able to read and understood Basic English language.
- Participants who score 18 or more in HAM-A

Exclusion criteria

- Age should not be more than 45 and less than 20 years
- Participants with other mental illness
- Participants with other medical illness

VARIABLES

Independent Variables

- Occupational therapy activities.

Dependent Variables

- Insomnia

MEASUREMENT TOOLS / MATERIALS USED

The tools used for the study is Pittsburgh insomnia rating scale and Hamilton Anxiety Scale.

DESCRIPTION OF THE INSTRUMENT

Pittsburgh insomnia rating scale: it is a 65 item self-administered open source questionnaire. PIRS is a widely used instrument in clinical and research practice. It is a scale with 65-items. It was designed to rate the severity of insomnia. Subjects score the items that have three broad sections.

1. Distress score (46 items)
2. Sleep parameters (10 items)
3. Quality-of-life (9 items).

Section A of the scale has a 10 cm line to mark the quality of sleep in the past week. This answer is not used in the scoring.[70]

Section B has 46 questions which have to be answered on the likert scale from 0-3

- 0 = not at all bothered,
- 1 = slightly bothered,
- 2 = moderately bothered,
- 3 = severely bothered.

Section C has 10 questions which have to be answered on the likert scale 0-3 with variable answers depending on the question. Score of this section is the addition of all and is termed as sleep parameters score. Section D has 9 questions which have to be answered on the likert scale from 0-3

- 0 = excellent,
- 1 = good,
- 2 = fair,
- 3 = poor.

Addition of all the answers gives the final score which is termed as Quality of life score. Section E is about comments which the patient wants to put in but it is not included in the scoring. Final score is the grand total of all the three components. Minimum score is 0 (good) and maximum is 195 (bad).

Reliability & Validity

The retest reliability for PIRS total score was 0.93 which indicates excellent reliability. Distress score ICC2 was 0.90 which indicates excellent reliability. Sleep parameters score ICC2 was 0.70 which indicates a good reliability. Quality of life score ICC2 was 0.71 which indicates a good reliability.

Concurrent validity: moderately positive correlation between PSQI and PIRS (r-0.31 P value-0.13), moderately positive correlation between PIRS and ISI (r-0.49 P value-0.012).

Internal consistency: Cronbach's alpha coefficient was calculated for Total PIRS score (α-0.93) which indicates excellent internal consistency. Component

score's Cronbach's alpha coefficient are- Distress score (α-0.82), Sleep parameters score (α-0.70) and Quality of life score (α-0.71) which is an acceptable internal consistency.

Homogeneity: PIRS total score to component scores ranged from high to moderately correlated positive relationship(r's-0.96 to 0.37) correlations between individual items and total score ranged from 0.07 to 0.96.

THE HAMILTON ANXIETY SCALE:

The HAM-A was one of the first rating scales developed to measure the severity of anxiety symptoms, and is still widely used today. The scale consists of 14 items, each defined by a series of symptoms, and measures both psychic anxiety (mental agitation and psychological distress) and somatic anxiety (physical complaints related to anxiety).

Each item is scored on a scale of 0 (not present) to 4 (severe), with a total score range of 0–56, where

- 14-17 = Mild Anxiety
- 18-24 = Moderate Anxiety
- 25-30 = Severe Anxiety

Reliability&Validity

The interrater reliability of HAM-A total score: 0.74; HAM-A total score of psychic anxiety: 0.73; HAM-A total score of somatic anxiety: 0.70. Items 10 (respiratory symptoms), 14 (behaviour at interview) and 13 (autonomic symptoms) had the lowest reliability (lower than 0.30). Concurrent validity of the HAM-A Total

scale score 0.75, Total subscale score (psychic anxiety) 0.80, Total subscale score (somatic anxiety) 0.85.

PROCEDURE

A total of 30 subjects were taken for this study. The subjects were selected from the age group between 20 to 45 years, already diagnosed as Insomnia and anxiety by a psychiatrist.Individuals who meets the selection criteria has been administrated with Hamilton Anxiety Rating Scale. Individual who score more than 18 in HAM-A are then selected and importance of the study has explained to them and obtained consent form each subjects. The subject then divided in to two groups; a control group and an experimental group, the control group consist of 15 subjects and also the experimental group, all the subjects were taken from Vivekananda health centre Erode.

The control group received only conventional therapies were as experimental group underwent occupational therapy intervention along with conventional therapy. Pre- test was conducted in both groups before the intervention with using Pittsburgh Insomnia Rating Scale

SESSIONS

The intervention spanned a period of eight weeks. Of these, there were seven weeks in which group occupational therapy sessions scheduled for participant along with 1:1 session (weeks 1, 2, 3, 4, 5, 6, and 8). Only 1:1 occupational therapy sessions were scheduledfor the remaining week (week 7). A group intervention approach was adopted for a principle reasons. A group intervention can be therapeutic via establishing the cohesiveness of the group, instilling hope, and allowing for

interpersonal learning.[30] Therefore, a group approach could allow for the development of a sense of comradery and peer support throughout program delivery. Secondly, group has demonstrated promise as an effective method for treating insomnia comorbid with mental health conditions .[30]The 1:1 occupational therapy was included within this because 1:1 intervention is the gold standard in the delivery treatment for chronic insomnia.[33]

Table 2

SESSIONS	CONTENT
1	Didactic education about: 1. Sleep architecture 2. Importance of sleep 3. Insomnia
2	Meditation: 1. Relaxation training 2. Mindful walking 3. Body scan
3	Stimulus control: 1. Association of bed with sleep. 2. If in bed for more than 10-15 minutes not sleeping, get out of bed, go to another room and do something enjoyable.Go back to bed only when sleepy. 3. No clock-watching. 4. Get out of bed within five minutes of alarm sounding.
4	Sleep Restriction Therapy (SRT) 1. Wake up at the same time every day (Decided together). 2. Go to bed at the same time every night. (Decided by occupational therapist based on average sleep diary data.) 3. No naps.

5	Sleep Hygiene 1. Keep room cool (20 degrees), quiet, and dark. 2. Limit alcohol and caffeine. 3. Quit using nicotine. 4. Exercise and eat healthy. 5. Keep daytime routine consistent. 6. Stay active/engaged throughout the day.
6	CBT: 1. Cognitive Restructuring
7	Individual session :addressing 1. Personal factors 2. Beliefs 3. Goal setting around stimulus control and sleep hygiene 4. Calculation of SE= (TST/TIB)×100
8	Role-Playing:

Table 3 : Pittsburgh insomnia rating scale

Subjects	Control group		Experimental group	
	Pre test	Post test	Pre test	Post test
1.	163	76	141	36
2.	162	90	156	74
3.	164	61	150	40
4.	158	67	149	30
5.	176	66	153	62
6.	165	64	161	51
7.	170	78	146	86
8.	163	149	175	76
9.	155	107	159	94
10.	147	107	175	61
11.	129	111	173	54
12.	135	112	163	66
13.	171	123	168	105
14.	125	109	161	55
15.	156	120	180	54

COMPARISON OF PRE TEST BETWEEN CONTROL AND EXPERIMENTAL GROUP

Table 4

Sl. No.	*PIR*	*Mean*	*Std. Error of Mean*	*S.D*	*"t" Value*	*"p" Value*
1	Pre-test (control)	155.9	3.974	15.39	0.9468	P>0.05
2	Pre-test (experimental)	160.7	3.033	11.75		

Graph 1

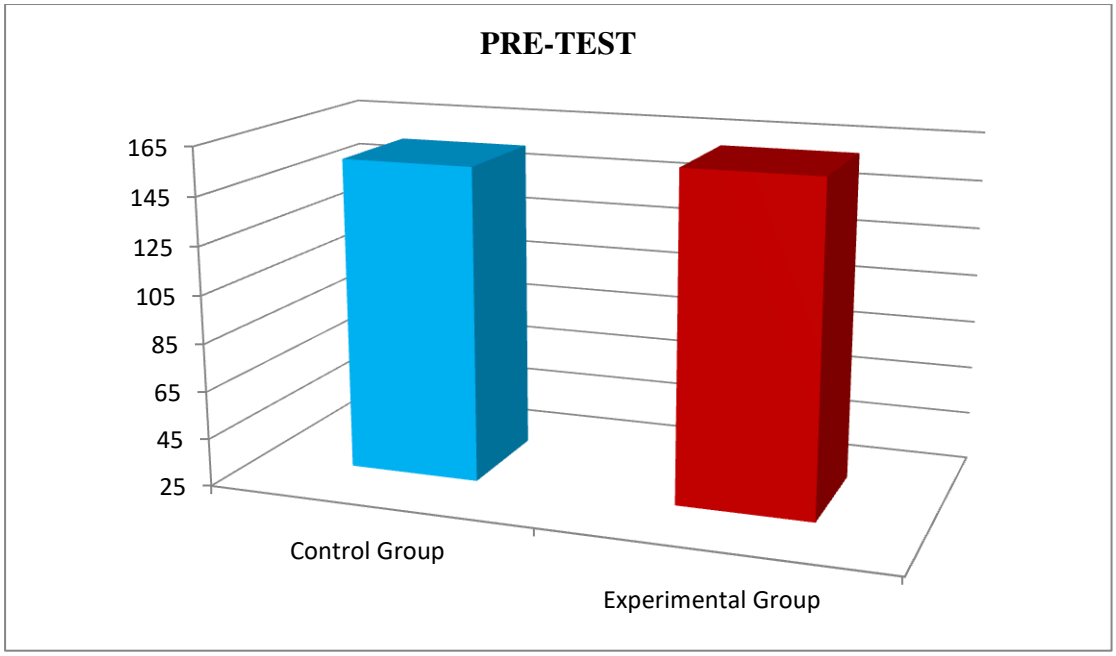

Table 4 shows that comparison between the control group Pre-test and experimental group Pre-test score mean values155.9;160.7 "t" values 0.9468 and "p" values 0.3518 which is greater than acceptance levels of significance of 0.05 so it is not statistically significant.

COMPARISONBETWEENPRE AND POST TESTSOF CONTROL GROUP

Table 5

Sl. No.	*PIR*	*Mean*	*Std. Error of Mean*	*S.D*	*"t" Value*	*"p" Value*
1	Pre-test	155.9	3.974	15.39	6.596	P<0.05
2	Post-test	96	6.816	26.4		

Graph 2

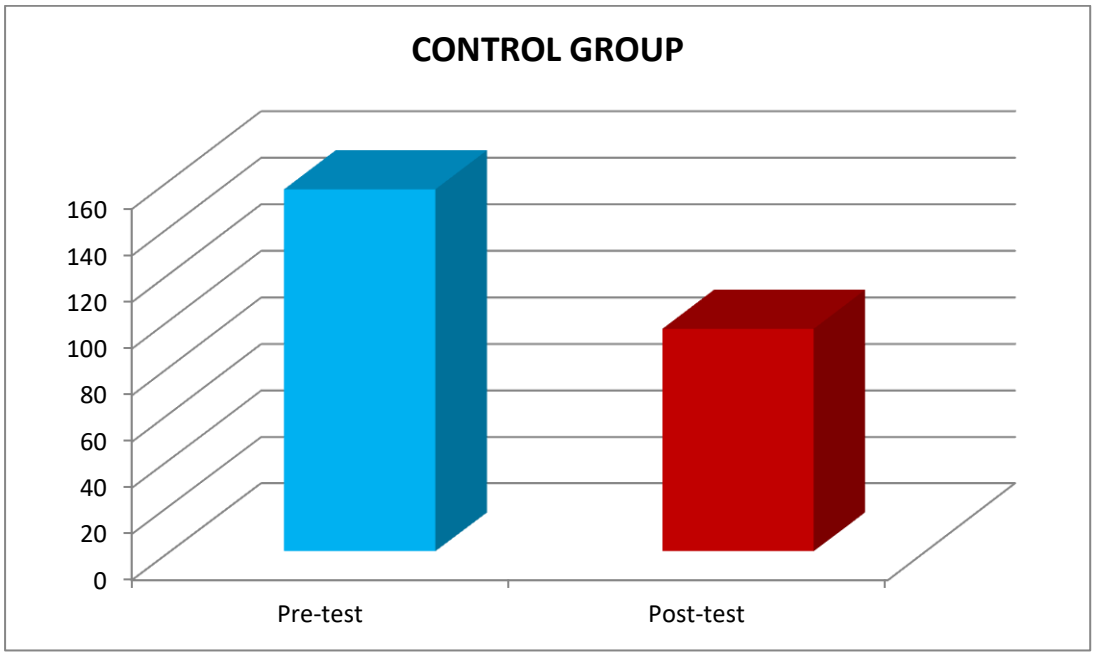

Table 5 shows that comparison between the control group pre-test and post test score mean values 155.9; 96 and "t" value 6.596 and "p" value which is Less than acceptance level of significance of 0.05. Hence it is statistically significant.

COMPARISON BETWEEN PRE AND POST TEST OF EXPERIMENTAL GROUP

Table 6

Sl.No.	*PIR*	*Mean*	*Std. Error of Mean*	*S.D*	*"t" Value*	*"p" Value*
1	Pre-test	160.7	3.033	11.75	17.72	P<0.05
2	Post-test	62.93	5.466	21.17		

Graph 3

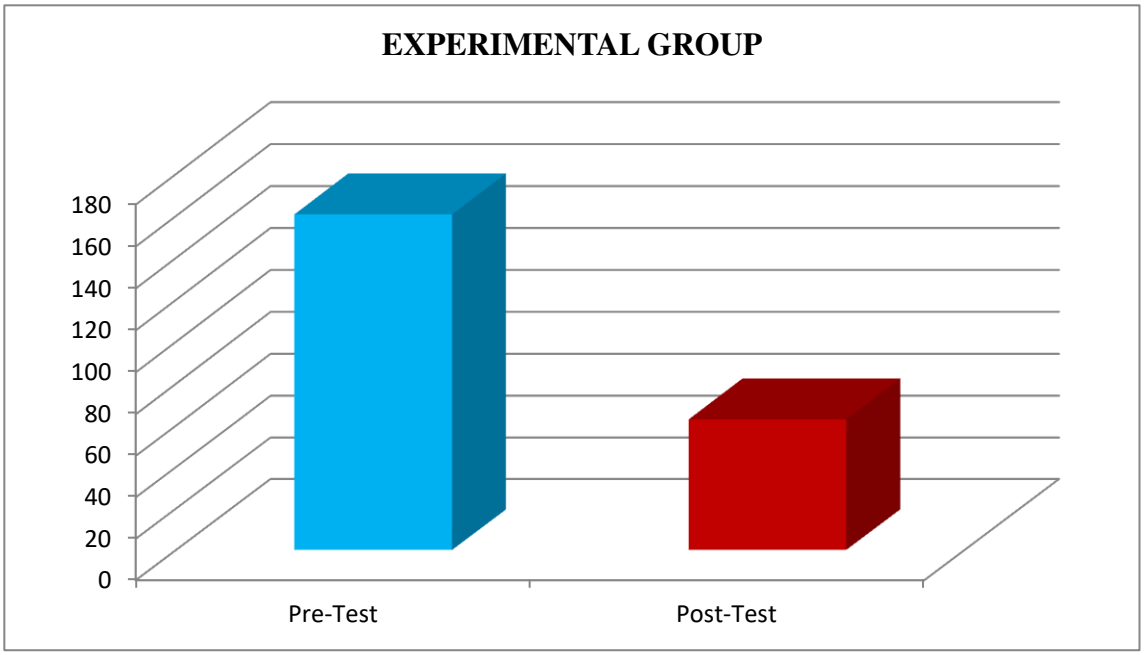

Table 6 shows that comparison between the experimental group pre-test and post test score mean values 160.7; 62.93and "t" values 17.72and "p" values which is less than acceptance level of significance of 0.05. Hence it is extremely statistically significant. It shows that experimental group has significant improvement.

COMPARISON BETWEEN POST TESTS OF CONTROL AND EXPERIMENTAL GROUP

Table 7

Sl. No.	*PIR*	*Mean*	*Std. Error of Mean*	*S.D*	*"t" Value*	*"p" Value*
1	Post-test (control)	96	6.816	26.4	3.785	P<0.05
2	Post-test (experimental)	62.93	5.466	21.17		

Graph 4

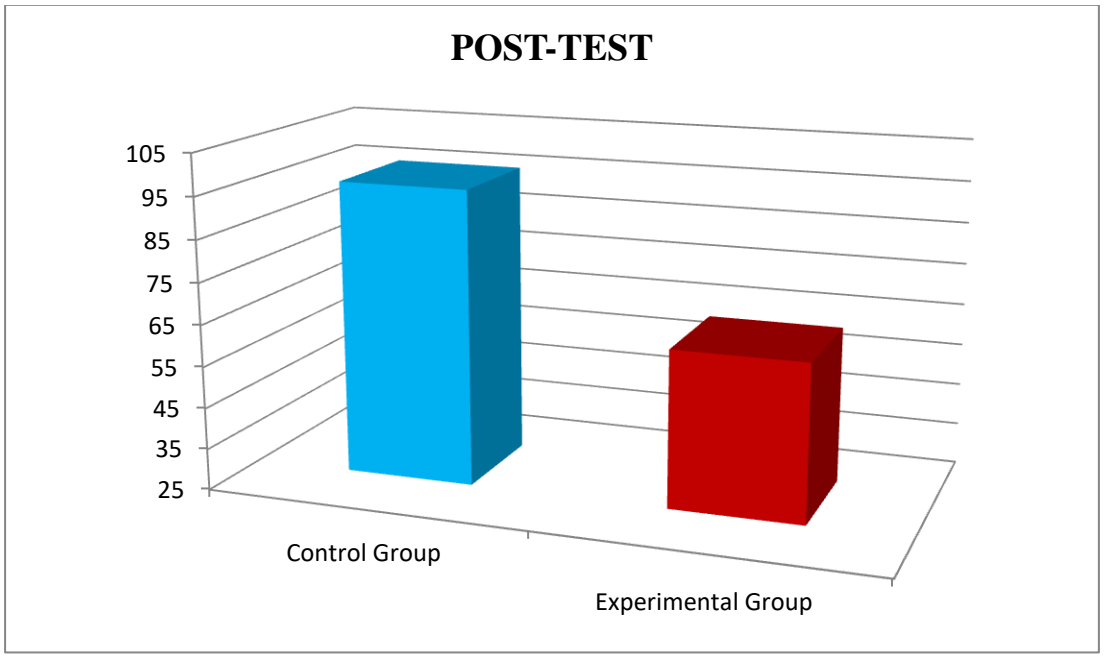

Table 7 shows that comparison between the control group post-test and experimental group post test score mean values 96; 62.93 "t" values 3.785 and "p" values which is less than acceptance levels of significance of 0.05. Hence it is statistically significant. It shows that experimental group has more improvement than that of control group.

DISCUSSION

Sleep disturbance is a clinical feature of nearly all psychiatric conditions and sleep is disrupted by symptoms of many physical conditions. Poor sleep totally affect the day time functioning of the person which leads to further complications. Occupational therapists could take a greater part in managing the sleep difficulties of their patients. It is proposed that from a more practical perspective there is a strong argument that they are well placed to assume a positive role. The purpose of this study is to find out effect of occupational therapy intervention in insomnia among person with anxiety.

The subjects for this study have been taken from Vivekananda health centre, individuals who meets the selection criteria has been administrated with Hamilton Anxiety Rating Scale. Individual who score more than 18 in HAM-A are invited to participate in the study and importance of the study has explained to them. Among those who was willing to participate in the study, consent form has taken and kept a cut off total 30 subjects as maximum sample size. Then the selected subjects has been allocated in two groups consist of 15 in each. The mean age of control group was 32.2 and 32 in Experimental group. Pre-test was done using Pittsburgh Insomnia Rating Scale after the pre-test experimental group received occupational therapy intervention for eight weeks along with conventional therapy and medication, whereas control group only receive conventional therapy and medication. After which a post- test was conducted using the same scale.

Unpaired ‘t’-test has been calculated for the both experimental and control group (Table 4). The mean of pre-test was found to be 155.9 (control) and 160.7

(experimental) respectively. The calculated 't' value was obtained (0.9468) with confidence level of 95%, P>0.05 which indicates there is no significant difference between both experimental and control group. Hence shows the homogeneity of the allocated subjects.

Paired 't' test has been calculated for the control group (Table 5). The mean of pre-test was found to be155.9 and the post test was 96. The calculated 't' value was obtained to be 6.596 with level of significance 0.05. Hence the table 't' value is less than calculated 't' value, which indicate significant difference between pre and post-test values.

This study did not include an active control condition thus, we cannot determine the effect of nonspecific factors, such as treatment expectancies and therapeutic alliance, socio economic factors and cultural influence.

Paired't' test have been calculated for the experimental group (Table 6). The mean of pre test was found to be 160.7 and the post test was 62.93. The calculated 't' value was obtained to be 17.72 with level of significance 0.05. Hence the table't' value is less than calculated 't' value. So there is extremely significant difference between pre and post-test values.

This is supported by Christi S. Ulmer etal (2011)[26] their study was Multi-Component Cognitive-Behavioural Intervention for Sleep Disturbance in Veterans with posttraumatic stress disorder and the subjects were twenty-two veterans. The findings demonstrate that an intervention targeting trauma-specific sleep disturbance produces large short-term effects, including substantial reductions in PTSD symptoms and insomnia severity.

Unpaired ‘t’ test has been calculated for the both experimental and control group (Table 7). The mean of post-test was found to be 96 (control) and 62.93 (experimental) respectively. The calculated‘t’ value was obtained to be 3.785 with level of significance 0.05. Hence the table‘t’ value is less than calculated ‘t’ value. So there is a significant difference between both group means, which leads to the rejection of null hypothesis.

This is supported by Aaron M Eakman et al (2017)[13] they conducted a pilot study on restoring effective sleep tranquillity (REST). REST was a multi-component cognitive behavioural therapy for insomnia intervention consisting of seven sessions of group therapy and eight 1:1 sessions delivered by occupational therapists. The indicators were supportive of feasibility, including reduced sleep difficulties, reduced nightmares,fewer dysfunctional sleep beliefs and greater ability to participate in social roles, along with trends towards improved satisfaction with participation and reduced pain interference.

CONCLUSION

The result of this study indicates that person who had occupational therapy intervention shows more improvement than who had conventional therapy. Therefore occupational therapy technique considered as a therapy intervention for Insomnia.

LIMITATION AND RECOMMENDATIONS

LIMITATION

- Study was done on a small sample size.
- Study was conducted in confined geographical location.
- Male and female comparison is not included in this study.
- Study was done on confined age group between 20 to 45 yrs

RECOMMENDATIONS

- The study can be done with other coexisting conditions.
- Study can be done by using electronic sleep monitoring devices.
- The study can be done by translating the scale into local language.
- Based on this study it is recommended to use occupational therapy interventions for person with insomnia.

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Hamilton Anxiety Rating Scale (HAM-A)

Below is a list of phrases that describe certain feeling that people have. Rate the patients by finding the answer which best describes the extent to which he/she has these conditions. Select one of the five responses for each of the fourteen questions.

0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe, 4 = Very severe.

1 Anxious mood [0] [1] [2] [3] [4]

Worries, anticipation of the worst, fearful anticipation, irritability.

2 Tension [0] [1] [2] [3] [4]

Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.

3 Fears [0] [1] [2] [3] [4]

Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.

4 Insomnia [0] [1] [2] [3] [4]

Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.

5 Intellectual [0] [1] [2] [3] [4]

Difficulty in concentration, poor memory.

6 Depressed mood [0] [1] [2] [3] [4]

Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.

7 Somatic (muscular) [0] [1] [2] [3] [4]

Pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.

8 Somatic (sensory) [0] [1] [2] [3] [4]

Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.

9 Cardiovascular symptoms [0] [1] [2] [3] [4]

Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.

10 Respiratory symptoms [0] [1] [2] [3] [4]

Pressure or constriction in chest, choking feelings, sighing, dyspnea.

11 Gastrointestinal symptoms [0] [1] [2] [3] [4]

Difficulty in swallowing, wind abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.

12 Genitourinary symptoms [0] [1] [2] [3] [4]

Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.

13 Autonomic symptoms [0] [1] [2] [3] [4]

Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.

14 Behavior at interview [0] [1] [2] [3] [4]

Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc.

Pittsburgh Insomnia Rating Scale

Name_____ ID#_____ Date_____

A. Overall sleep quality: Consider the quality of your sleep in the past 7 days. Then mark that point along the line that best describes your sleep quality in the past 7 days:

Horrible |——————————————————————| Wonderful

*The following questions ask about your sleep **in the past 7 days and nights**. Please circle the one **best** answer for each question.*

B. In the past week, how much were you bothered by:	**Not at all bothered**	**Slightly bothered**	**Moderately bothered**	**Severely bothered**
1. Difficulty getting to sleep at bedtime	0	1	2	3
2. One or more awakenings after getting to sleep	0	1	2	3
3. Waking up too early in the morning	0	1	2	3
4. Not getting enough sleep	0	1	2	3
5. Different sleep patterns from one night to the next	0	1	2	3
6. Sleep occurring at odd times or not at all	0	1	2	3
7. Intense or disturbing dreams	0	1	2	3
8. Sensations (like noises, hot or cold, pain) during the night	0	1	2	3
9. Physical tension at night	0	1	2	3
10. Moving too much in bed	0	1	2	3
11. Anxiety or worries about getting to sleep	0	1	2	3
12. Anxiety or worries about lack of sleep	0	1	2	3
13. Anxiety or worries about what might happen during sleep	0	1	2	3
14. General nervousness and stress	0	1	2	3
15. Poor sleeping causing you to feel stress	0	1	2	3
16. Stress causing poor sleeping	0	1	2	3
17. Your mind not slowing down at bedtime	0	1	2	3

In the past week, how much were you bothered by:	Not at all bothered	Slightly bothered	Moderately bothered	Severely bothered
18. Loss of desire for physical intimacy or sex	0	1	2	3
19. Sleep that doesn't fully refresh you	0	1	2	3
20. Difficulty waking up	0	1	2	3
21. Poor alertness during the daytime	0	1	2	3
22. Difficulty keeping your thoughts focused	0	1	2	3
23. Your mind never slowing down during the daytime	0	1	2	3
24. Difficulty remembering things	0	1	2	3
25. Difficulty thinking clearly and making decisions	0	1	2	3
26. Tiredness or fatigue	0	1	2	3
27. Dozing off or napping when you really didn't want to	0	1	2	3
28. Others noticing you appeared tired or fatigued	0	1	2	3
29. Too many difficulties to overcome	0	1	2	3
30. Being unsure about handling your personal problems	0	1	2	3
31. Being unsure about dealing with day-to-day problems	0	1	2	3
32. Irritation with sounds, sights, or sensations during the day	0	1	2	3
33. Bad mood(s) because you had poor sleep	0	1	2	3
34. Irritation with people even when they were polite	0	1	2	3
35. Difficulty controlling your emotions	0	1	2	3
36. Needing to keep quiet around other people	0	1	2	3
37. Lack of energy because of poor sleep	0	1	2	3
38. Poor sleep that interferes with your relationships	0	1	2	3
39. Feeling sleepy	0	1	2	3
40. Being unable to sleep	0	1	2	3
41. Feeling that time itself slowed down	0	1	2	3
42. Being able to do only enough to get by	0	1	2	3
43. Difficulty getting along with other people	0	1	2	3
44. Physical clumsiness	0	1	2	3
45. Feeling physically ill or prone to infections	0	1	2	3
46. Being forced to pay special attention to what you eat or what you do so that you can sleep better	0	1	2	3

C. Please circle the best answer for each question about the past week:

47. From the time you tried to go to sleep, how long did it take to fall asleep on the **worst** night?
    0 Less than ½ hour
    1 Between ½ to 1 hour
    2 Between 1 to 3 hours
    3 More than 3 hours or I didn't sleep.

48. From the time you tried to go to sleep, how long did it take to fall asleep on **most** nights?
    0 Less than ½ hour
    1 Between ½ to 1 hour
    2 Between 1 to 3 hours
    3 More than 3 hours or I didn't sleep.

49. If you woke up during the night, how long did it take to fall back to sleep on the **worst** night?
    0 Less than ½ hour or I didn't wake up
    1 Between ½ to 1 hour
    2 Between 1 to 3 hours.
    3 More than 3 hours or I didn't fall back to sleep.

50. If you woke up during the night, how long did it take to fall back to sleep on **most** nights?
    0 Less than ½ hour or I didn't wake up
    1 Between ½ to 1 hour
    2 Between 1 to 3 hours.
    3 More than 3 hours or I didn't fall back to sleep.

51. Not counting times when you were awake in bed, how many hours of **actual** sleep did you get during the **worst** night?
    0 More than 7 hours.
    1 Between 4 to 7 hours.
    2 Between 2 to 4 hours.
    3 Less than 2 hours or I didn't sleep.

52. Not counting times when you were awake in bed, how many hours of **actual** sleep did you get during **most** nights?
    0 More than 7 hours
    1 Between 4 to 7 hours
    2 Between 2 to 4 hours
    3 Less than 2 hours or I didn't sleep.

53. On how many nights did it take **longer** than 30 minutes to fall to sleep?
    0 None or 1 night
    1 On 2 or 3 nights
    2 On 4 or 5 nights
    3 On 6 or all nights

54. On how many nights did you wake up and have **trouble falling back** to sleep?
    0 None or 1 night
    1 On 2 or 3 nights
    2 On 4 or 5 nights
    3 On 6 or all nights

55. On how many mornings did you wake up **not fully rested**?
    0 None or 1 morning
    1 On 2 or 3 mornings
    2 On 4 or 5 mornings
    3 On 6 or all mornings

56. On how many days did you have trouble coping **because of poor sleep**?
    0 None or 1 day
    1 On 2 or 3 days
    2 On 4 or 5 days
    3 On 6 or all days

D. Over the past week, how would you rate:	Excellent	Good	Fair	Poor
57. Your sleep quality, compared to most people	0	1	2	3
58. Your satisfaction with your sleep	0	1	2	3
59. Your ability to get things done, compared to your best ..	0	1	2	3
60. Your satisfaction with how you got things done	0	1	2	3
61. The regularity of your sleep	0	1	2	3
62. The soundness of your sleep	0	1	2	3
63. How well you talked and communicated with others	0	1	2	3
64. Your sense of humor	0	1	2	3
65. Your quality of life	0	1	2	3

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Rtn.MPHF. **Dr.J.K.K. MUNIRAJAHH** M.Tech., (Bolton)
Correspondent

MOT/Project-Permission/2017

Date : 19.08.2017

To
Vivekananda Health Centre,
209/20-A, Tamil Nagar,
Karur Bye Pass Road,
Erode - 638 002.

Respected Sir/Madam,

Sub: Regarding permission to project data collection.

* * *

With reference to the subject cited above, our Master of Occupational Therapy Second year student **RICHU JOSEPH** is doing project on the topic "Effect of Occupational Therapy Interventions in Insomnia among patients with Anxiety". He likes to collect data from your centre. So, we request you to give permission for the above student to collect the data for his project.

Thanking you,

Yours sincerely,

PRINCIPAL
JKKMMRF COLLEGE OF
OCCUPATIONAL THERAPY
KOMARAPALAYAM - 638 18?

Permission granted
Saravanan.

Dr. E.S.M. SARAVANAN,
M.B.B.S., D.P.M., DNB (Psychiatry)
Reg No. 55095

MASTER CHART

Subject code	Age	Sex	Pittsburgh Insomnia Rating Scale		HAM-A
			Pre-test	Post-test	
1.	38	M	141	36	26
2.	43	M	156	74	34
3.	30	M	150	40	30
4.	40	F	149	30	27
5.	32	F	153	62	36
6.	22	M	161	51	34
7.	29	F	146	86	30
8.	31	F	175	149	39
9.	29	F	159	107	28
10.	28	M	175	107	34
11.	27	M	173	111	41
12.	38	M	163	112	32
13.	41	M	168	123	30
14.	29	M	161	109	23
15.	27	F	180	120	40
16.	32	F	163	76	29
17.	30	F	162	90	32
18.	41	F	164	61	35
19.	34	F	158	67	30
20.	28	F	176	66	34
21.	39	M	165	64	26
22.	36	M	170	78	47
23.	29	M	163	76	34
24.	43	M	155	94	29
25.	29	M	147	61	27
26.	26	M	129	54	23
27.	29	M	135	66	24
28.	27	F	171	105	42
29.	28	M	125	55	19
30.	29	M	156	54	25

CONFIDENTIALITY

Your name will not be associated with the results in this study. It will be issued for both teaching and research purpose. Only myself and my guide will have access to the name of the subjects participating in this study.

The following is the name address and telephone number of the person to be contacted in event of research related inquiry.

Name : Richu Joseph

Address : JKKMMRF College of Occupational Therapy

Komarapalayam, Namakkal Dt.

VOLUNTARY CONCERN FORM

NAME :

AGE :

SEX :

ADDRESS FOR COMMUNICATION :

DECLARATION

I have fully understood the nature and purpose of the study. I have understood that I have the right to withdraw from this study at any point of time without adversely affect my treatment. I have read this consent form and accept to be a subject in this study on my own accord. I declare that the above information is true to my knowledge.

Signature of the informant

Date:

Place: